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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/051,497	01/18/2002	Rong-Hwa Lin	A0871.70000US01	1774

23628 7590 07/31/2006

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EXAMINER	
GAMBEL, PHILLIP	

ART UNIT	PAPER NUMBER
1644	

DATE MAILED: 07/31/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/051,497	LIN ET AL.	
	Examiner	Art Unit	
	Phillip Gambel	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *30 May 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,3,4,6-13,17,19 and 22-25* is/are pending in the application.

4a) Of the above claim(s) *7-9* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,3,4,6,10-13,17,19,20 and 22-25* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114.

Applicant's submission, filed on 5/30/06, has been entered.

Applicant's amendment, filed 5/30/06, has been entered.
Claims 1, 3, 17, 19 and 25 have been amended.

Claims 2, 5, 14-16, 18, 21, and 26-37 have been canceled previously.

Claims 1, 3, 4, 6-13, 17, 19, 20 and 22-25 are pending.

As pointed out previously, applicant's election of species (B), drawn to methods using an anti-PSGL-1 antibody and an agent that binds to the antibody and induces the cross-linking of a plurality of PSGL-1 antigens on the cell surface without traverse in the Reply, filed 7/22/04, and the species autoimmune disease and type I diabetes in the Reply, filed 3/10/04, has been acknowledged.

Claims 1, 3, 4, 6, 10-13, 17, 19, 20, 22, 23, 24 and 25 are under consideration in the instant application.

Claims 7-9 have been withdrawn from consideration by the examiner 37 CFR 1.142(b), as being drawn to a nonelected invention or species.

2. The text of those sections of Title 35 USC not included in this Action can be found in a prior Action.

This Action will be in response to applicant's amendment, filed 5/30/06.
The rejections of record can be found in the previous Office Action.

3. As indicated previously, the filing date of the instant claims is deemed to be the filing date of the instant application USSN 10/051,497, filed 1/18/02;
as the previous provisional priority application USSN 60/310,196, filed 8/3/01, does not appear to provide sufficient written description for the claimed "limitations".

Applicant's assertions, filed 5/30/06, concerning the priority of the inst[illegible]
back to priority USSN 60/310,196, filed 8/3/01, are acknowledg[illegible]

However, these assertions are not found convincing essentia[illegible]
record reiterated herein for applicant's convenience.

As indicated previously, the instant claims now recite limitations which were not clearly disclosed in the priority provisional application as well as the specification as-filed, and would have changed the scope of the priority application and do change the scope of the instant disclosure as-filed.

For example, it cannot be said that a subgenus is necessarily described by a genus encompassing it and a species upon which it reads. See In re Smith 173 USPQ 679, 683 (CCPA 1972) and MPEP 2163.05.

Therefore, applicant's reliance on generic methods to reduce T cell-mediated immune responses with PSGL-1-specific antibodies and certain limitations found in the Examples of the provisional application does not provide sufficient written description for the claimed limitations indicated previously and herein, as currently claimed.

As indicated previously, the filing date of the instant claims as they read on "methods of preventing or reducing a T cell-mediated immune responses in an individual, including the "selecting an individual diagnosed", "administering a compound ... induces a signal transduction pathway that results in the death of the T cell" (e.g. claim 1), "an agent that binds to the monoclonal antibody and induces the cross-linking of a plurality of PSGL-1 antigens on the surface of the T cell" (e.g. claim 4), detecting the number of T cells in a first biological sample (e.g. claims 13-14), "20% of peripheral blood $CD3^+$ cells (e.g. claims 15-16) and "diabetes" (e.g. elected autoimmune disease) is deemed to be the filing date of the instant application USSN 10/051,497, filed 8/3/01, as the previous provisional priority application does not appear to provide sufficient written description for the claimed "limitations" indicated herein.

Although applicant disagrees with this analysis, applicant has not presented a detailed analysis as to why the claimed subject matter has clear support in the parent application, other than to assert that the provisional application provides ample written description for each and every limitation as presented and citing certain passages of the provisional application without sufficiently pointing out written support for the "limitations" indicated previously and herein.

Again, applicant is invited to verify the priority date of the instant claims, including written support and enablement under 35 USC 112, first paragraph.

Again, if applicant disagrees, applicant should present a detailed analysis as to why the claimed subject matter has clear support in the parent application. Applicant is invited to verify the priority date of the instant claims, including written support and enablement under 35 USC 112, first paragraph.

4. This is a rejection under 35 USC § 112, first paragraph, "new matter".

Claims 4 and 20 are rejected under 35 U.S.C. § 112, first paragraph, as the specification does not contain a written description of the claimed invention, in that the disclosure does not reasonably convey to one skilled in the relevant art that the inventor(s) had possession of the claimed invention at the time the application was filed.

The specification as originally filed does not provide support for the invention as now claimed:

"an antibody that binds to the monoclonal antibody and induces the cross-linking of a plurality of PSGL-1 antigen on the surface of the T cell".

Applicant's arguments, filed 5/30/06, have been fully considered but have not been found convincing essentially for the reasons of record.

Again, it appears that applicant is relying upon the disclosure of the anti-hamster Ig as a cross-linker antibody (e.g. see Example 3 on pages 19-20) to support an entire (sub)genus of "antibodies that bind to the monoclonal antibody and induces the cross-linking of a plurality of PSGL-1 antigen on the surface of the T cell".

Other than the reliance upon the Example 3,

the specification discloses "agents that bind to the monoclonal antibody and induces the cross-linking of a plurality of PSGL-1 antigens on the surface of cell" (e.g. see Summary of the Invention, particularly page 5, paragraph 3 of the instant specification).

Again, and consistent with applicant's arguments;

applicant's reliance on a generic disclosure (e.g. agent) and possibly a single or limited species (e.g. anti-hamster Ig in an Example) does not provide sufficient direction and guidance to the generic "antibody that binds to the monoclonal antibody and induces the cross-linking of a plurality of PSGL-1 antigen on the surface of the T cell", as currently claimed.

It is noted that a generic or a sub-generic disclosure cannot support a species unless the species is specifically described. It cannot be said that a subgenus is necessarily described by a genus encompassing it and a species upon which it reads. See In re Smith 173 USPQ 679, 683 (CCPA 1972) and MPEP 2163.05.

Applicant relies upon the ordinary artisan recognizing the possession of a subgenus of cross-linking antibodies based upon Example 3, rather than clear written description of the claimed "limitation" in the application as filed.

The specification as filed does not provide a sufficient written description nor provide sufficient blazemarks nor direction for the instant methods encompassing the above-mentioned "limitation", as currently recited. The instant claims now recite limitations which were not clearly disclosed in the specification as-filed, and now change the scope of the instant disclosure as-filed. Such limitations recited in the present claims, which did not appear in the specification, as filed, introduce new concepts and violate the description requirement of the first paragraph of 35 U.S.C. 112.

Applicant's arguments have not been found persuasive.

Applicant is required to cancel the new matter in the response to this Office Action.

Alternatively, applicant is invited to provide sufficient written support for the "limitation" indicated above.
See MPEP 714.02 and 2163.06

5. Claims 4 and 20 are rejected under 35 U.S.C. § 112, first paragraph, because the specification, while being enabling "anti-hamster Ig" and "rabbit anti-mouse Ig" as "an antibody that bind to the monoclonal antibody and induces the cross-linking of a plurality of PSGL-1 antigens on the surface of cell",
does not reasonably provide enablement for any "antibody that binds to the monoclonal antibody and induces the cross-linking of a plurality of PSGL-1 antigens on the surface of T cell".
The specification does not enable any person skilled in the art to which it pertains, or with which it is most clearly connected, to make and use the invention commensurate in scope with these claims.

Applicant's arguments, in conjunction with certain references, have been fully considered but are not found convincing essentially for the reasons of record.

Applicant argues in conjunction with the references submitted on the Supplemental IDS that the teachings of the present application and the state of the prior art, the skilled artisan could obtain a cross-linking antibody as recited in the claims without undue experimentation.

However, applicant has not provided sufficient direction and guidance in the specification as filed as how to make and to use such cross-linking antibodies in the claimed methods, as generically claimed.

Again as applicant acknowledge, applicant appears to be relying upon the disclosure of the anti-hamster Ig or rabbit anti-mouse Ig as a cross-linker antibody (e.g. see Example 3 on pages 19-20 and Example 10 on pages 26-27) to support an entire genus of "antibodies that bind to an anti-PSGL-1 antibody".

This invention encompasses any "antibody that binds to the monoclonal antibody and induces the cross-linking of a plurality of PSGL-1 antigens on the surface of cell" (e.g. see Summary of the Invention, particularly page 5, paragraph 3 of the instant specification), yet the instant specification does not provide sufficient direction and guidance as to the nature of antibodies that can induce cross-linking in vivo, broadly encompassed by the claimed invention.

While cross-linking antibodies in vivo may be accomplished by various antibody constructs, including multimeric antibodies, or antibodies that bind anti-PSGL antibodies that are not hamster antibodies, the instant disclosure provides for insufficient guidance and direction towards the relevant, identifying characteristics of the "antibodies that bind to an anti-PSGL-1 antibody and induces the cross-linking of a plurality of PSGL-1 antigens on the surface of T cell".

Reasonable correlation must exist between the scope of the claims and scope of enablement set forth. The specification does not describe nor enable any "antibody that binds to the monoclonal antibody and induces the cross-linking of a plurality of PSGL-1 antigens on the surface of T cell, other than "anti-hamster antibody".

Without sufficient guidance, making and using an "antibody that binds to the monoclonal antibody and induces the cross-linking of plurality of PSGL-1 antigens on the surface of T cell" other than the "anti-hamster/mouse Ig" disclosed in the specification as filed as the "antibody" in the claimed methods would have been unpredictable and the experimentation left to those skilled in the art is unnecessarily, and improperly, extensive and undue.

Applicant's arguments have not been found persuasive.

6. For clarity and convenience and as pointed out previously,
for examination purposes, it appears that claimed methods which rely upon the elected species of administering an anti-PSGL-1 antibody and an "agent that binds to the monoclonal antibody and induces the cross-linking of plurality of PSGL-1 antigens on the surface of T cell appears free of the prior art.

Accordingly, the prior art rejections have been extended to read on the species of administering anti-PSGL-1 antibodies in the absence of administering a secondary "cross-linking agent".

As indicated in the prior art rejections of record, it does not appear that the claim language or limitations result in a manipulative difference in the method steps when compared to the prior art disclosures of administering anti-PSGL-1 antibodies to inhibit P-selectin- / PSGL-1-mediated interactions, including immune responses such as the elected autoimmune disease diabetes.

However, the prior art disclosures are silent on the claimed recitation of "inducing a signal transduction pathway that results in the death of a T cell or NK cell".

As pointed out above, the Office is not equipped to conduct comparisons. Applicant is invited to provide clarity and objective evidence as to the mechanism of action by which the administration of anti-PSGL-1 antibodies can reduce T cell mediated immune responses, including autoimmunity such as diabetes in the absence of a secondary cross-linking agent.

Also, as noted herein, co-inventors own publication Chen et al. (Blood 104: 3233-3242, 2004) indicates that PSGL-1 mediated death via PSGL-1-specific antibodies is stage dependent in that it affects mature activated T cells (see entire document, particularly the Discussion).

Therefore, applicant's reliance on "inducing a signal transduction that results in the death of the T cell thereby reducing a T cell-mediated immune response in the individual" appears based not on the nature of the anti-PSGL-1 antibody but rather based on the presence of PSGL-1 expressing mature activated T cells.

7. Claims 1, 3, 6, 10-12, 17, 19 and 22-24 are rejected under 35 U.S.C. § 102(b) as being anticipated by Larsen et al. (U.S. Patent No. 5,840,679) (see entire document) essentially for the reasons of record and in further evidence of Chen et al. (Blood 104: 3233-3242, 2004).

Applicant' arguments, filed 5/30/06, have been fully considered but are not found convincing essentially for the reasons of record.

Applicant submits that not all anti-PSGL-1 antibodies can induce T cell apoptosis.

While applicant asserts that various candidate anti-PSGL antibodies cannot induce death of activated T cells, no evidence has been provided to support these observations.

Also, in the absence of such information, it is difficult to ascertain the distinctions between the claimed and prior art methods, which inhibit T cell-mediated immune responses with PSGL-1-specific antibodies.

Further, co-inventors own publication Chen et al. (Blood 104: 3233-3242, 2004) indicates that PSGL-1 mediated death via PSGL-1-specific antibodies is stage dependent in that it affects mature activated T cells (see entire document, particularly the Discussion).

Therefore, applicant's reliance on "inducing a signal transduction that results in the death of the T cell thereby reducing a T cell-mediated immune response in the individual" appears based not on the nature of the anti-PSGL-1 antibody but rather based on the presence of PSGL-1 expressing mature activated T cells.

Applicant has previously argued that the prior art is drawn to the use of antagonistic anti-PSGL-1 antibodies, while the instant claims are drawn to the use of agonistic antibodies.

Again, neither the claims nor the specification make it clear that the instant antibodies are necessarily agonistic antibodies and agonistic in terms of what endpoints, while the prior art taught antagonistic antibodies.

As noted by co-inventors own publication Chen et al. (Blood 104: 3233-3242, 2004), the issue appears not to be one of agonistic or antagonistic antibodies but rather the presence of PSGL-1 expressing mature activated T cells during the administration of PSGL-1-specific antibodies.

It is unclear how applicant is making this distinction, given that the claims rely upon anti-PSGL antibodies with no mention of agonistic properties and both the instant and prior art anti-PSLG-1 antibodies are administered to inhibit T cell mediated immune responses.

The reduction of T cell mediated immune responses may occur via multiple modes of action.

While applicant relies upon in vitro analysis and cross-linking of anti-PSGL antibodies under in vitro experimental conditions to show evidence of apoptosis, applicant does not provide sufficient objective evidence that the administration of anti-PSGL antibodies in vivo does or does not require cross-linking to achieve the decrease in T cell numbers and T cell mediated immune responses in view of antibody treatment of an experimental autoimmune diabetes model (see instant Example 11).

While the invention may be based on the discovery that T cells can be depleted and/or induced to undergo apoptosis by the engagement of the T cell surface antigen PSGL (e.g. Summary of the Invention and applicant's arguments),
there is insufficient objective evidence that the treatment of anti-PSGL-1 antibodies in the prior art do not result in the claimed cell death of T cells via cross-linking (e.g. via Fc- FcR binding) and/or the presence of PSGL-1 expressing mature activated T cells during the administration of PSGL-1-specific antibodies.

Also, while Example 11 discloses the use of anti-PSGL-1 antibodies to treat an experimental model of diabetes, there is insufficient objective evidence that the reduction of T cell mediated immune response is accomplished via the death of the T cells and does not rely upon other mechanisms in vivo.

Although the reference is silent about the induction of T cell or NK cell death as well as identifying the T cell as activated, $CD3^+$, $CD4^+$, or $CD8^+$ as well as the depletion of T cells, it does not appear that the claim language or limitations result in a manipulative difference in the method steps when compared to the prior art disclosure.

The products employed in the instant methods and the prior art are defined in terms of anti-PSGL-1 antibodies. Comparison of the instant products with prior art is difficult since the Office is not equipped to manufacture the claimed product and/or prior art products that appear to be related and conduct comparisons

Applicant is invited to clarify the distinctions between the properties of anti-PSGL-1 antibodies and the instant anti-PSGL-1 antibodies and claimed the antibodies accordingly.

At this time, it is difficult for the examiner to determine
whether applicant has discovered a new mode of action (e.g. induction of apoptosis) of anti-PSGL-1 antibodies,
whether applicant has discovered a new epitope specificity of apoptosis-inducing anti-PSGL-1 antibodies,
whether applicant is relying upon secondary cross-linking agents / antibodies to accomplish this newly discovered mode of action and/or
whether the administration of anti-PSGL-1 antibodies even in applicant's model operate via apoptosis in vivo in the absence of secondary cross-linking agents / antibodies.

As indicated herein, applicant's reliance on "inducing a signal transduction that results in the death of the T cell thereby reducing a T cell-mediated immune response in the individual" appears based not on the nature of the anti-PSGL-1 antibody or the nature of the specificity of the anti-PSGL-1 antibody but rather based on the presence of PSGL-1 expressing mature activated T cells during the administration of PSGL-1-specific antibodies.

Therefore the prior art stands at this time, given it does not appear that the claim language or limitations result in a manipulative difference in the method steps when compared to the prior art disclosure.

See the previous Office Action for a more complete analysis of the prior art rejection.

8. Claims 1, 3, 6, 10-13, 19, 20, 22, 23, 24 and 25 are rejected under 35 U.S.C. § 103(a) as being unpatentable over by Larsen et al. (U.S. Patent No. 5,840,679) in view of Trembleau et al. (J. Immunol. 163 : 2960 – 2968, 1999), Yago et al. (J. Immunol. 161 : 1140 –1145 (1998), Hirata et al. (J. Exp. Med. 192: 1669 – 1675, 2000) and Cobbold et al. (U.S. Patent No. 6,056,956) and as futher evidenced by Chen et al. (Blood 104: 3233-3242, 2004) essentially for the reasons of record.

Applicant's arguments, filed 5/30/06, and the examiner's rebuttal are essentially the same set forth above.

Therefore, applicant's arguments have been fully considered but are not found convincing essentially for the reasons of record.

See the previous Office Action for a more complete analysis of the prior art rejection.

While the invention may be based on the discovery that T cells can be depleted and/or induced to undergo apoptosis by the engagement of the T cell surface antigen PSGL (e.g. Summary of the Invention and applicant's arguments),
there is insufficient objective evidence that the treatment of anti-PSGL-1 antibodies in the prior art do not result in the claimed cell death of T cells via cross-linking (e.g. via Fc- FcR binding) and/or the presence of PSGL-1 expressing mature activated T cells during the administration of PSGL-1-specific antibodies.

Applicant's arguments have not been found persuasive.

9. The non-statutory double patenting rejection, whether of the obvious-type or non-obvious-type, is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent. In re Thorington, 418 F.2d 528, 163 USPQ 644 (CCPA 1969); In re Vogel, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); In re Van Ornam, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); In re Longi, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); and In re Goodman, 29 USPQ2d 2010 (Fed. Cir. 1993).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321 (b) and (c) may be used to overcome an actual or provisional rejection based on a non-statutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.78 (d).

Effective January 1, 1994, a registered attorney or agent of record may sign a Terminal Disclaimer. A Terminal Disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

10. Claims 1, 3, 4, 6, 10-13, 17, 19, 20, 22, 23, 24 and 25 are provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1, 4, 8, 9, 12-15, 19-22, 23, 26, 30, 31, 34-38 of copending application USSN 10/662,906. Although the conflicting claims are not identical, they are not patentably distinct from each other because they are drawn to the same or nearly the same methods of preventing or reducing T cell-mediated immune responses with the same nearly the same PSGL-1-specific antibodies. Therefore, the copending claims either anticipate or render obvious one another.

It is noted that the copending claims recite a "multimeric compound that binds at least two PSGL-1 proteins". Given that the copending claims also recite "anti-PSGL-1 antibodies" and that antibodies have two binding sites, the copending claims appear to read on the instant claims drawn to the essentially the same methods relying upon PSGL-1 antibodies.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

11. Claims 1, 3, 4, 6, 10-13, 17, 19, 20, 22, 23, 24 and 25 are directed to an invention not patentably distinct from claims 1, 4, 8, 9, 12-15, 19-22, 23, 26, 30, 31, 34-38 of commonly assigned USSN 10/662,906 for the reasons set forth above.

The U.S. Patent and Trademark Office normally will not institute an interference between applications or a patent and an application of common ownership (see MPEP § 2302). Commonly assigned USSN 10/662,906, discussed above, would form the basis for a rejection of the noted claims under 35 U.S.C. 103(a) if the commonly assigned case qualifies as prior art under 35 U.S.C. 102(e), (f) or (g) and the conflicting inventions were not commonly owned at the time the invention in this application was made. In order for the examiner to resolve this issue, the assignee can, under 35 U.S.C. 103(c) and 37 CFR 1.78(c), either show that the conflicting inventions were commonly owned at the time the invention in this application was made, or name the prior inventor of the conflicting subject matter.

A showing that the inventions were commonly owned at the time the invention in this application was made will preclude a rejection under 35 U.S.C. 103(a) based upon the commonly assigned case as a reference under 35 U.S.C. 102(f) or (g), or 35 U.S.C. 102(e) for applications pending on or after December 10, 2004.

12. No claim allowed.

13. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Phillip Gambel whose telephone number is (571) 272-0844. The examiner can normally be reached Monday through Thursday from 7:30 am to 6:00 pm. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christina Chan can be reached on (571) 272-0841.

The fax number for the organization where this application or proceeding is assigned is 571-272-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

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July 19, 2006